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Maternal obesity: gender-specific differences in placental pathology

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resistance in vitro. The research was to investigate whether accumulation of plasma and placental AOPP were associated with placenta-mediated pregnancy complications(PMPC) in gestational diabetes mellitus (GDM).
Methods: Fifty-three GDM patients, including 24 with and 29 without PMPC were enrolled. Thirty healthy singleton pregnant women were selected as control during March 1st,2010 and January 1st,2012 in Nanfang Hospital, Guangzhou, China. PMPC included preeclampsia(n=15), intrauterine growth restriction(n=3), intrauterine fetal demise(n=6). Plasma samples were collected at admission before delivery and approximately 42 days postpartum. Placental samples were obtained immediately after delivery from 23 normal pregnancies, 24 and 23 GDM patients with or without PMPC. We measured plasma levels of AOPP, glucose, lipid, and hormone profiles. The clinical features and the laboratory parameters were analyzed. Pearson correlation was performed to evaluate the associations between plasma AOPP level and glucose, lipid profiles as well as insulin resistance.
Results: The mean plasma AOPP levels were significantly different among normal pregnancies, GDM without or with PMPC (48.7 ± 8.2μmol/L, 63.5 ± 13.1μmol/L and 89.6 ± 10.8μmol/L). Higher levels of placental AOPP were observed in GDM with PMPC when compared to normal pregnancies and GDM without PMPC (46.8 ± 6.5μmol/mg protein, 16.4 ± 5.6μmol/mg protein and 25.8 ± 6.1μmol/mg protein). Pearson correlation analysis demonstrated plasma AOPP were positively correlated with placental AOPP ($r = 0.82$; $P<.01$), fasting glucose ($r = 0.41$; $P<.01$), A1C ($r = 0.65$; $P<.01$), triglyceride ($r = 0.38$; $P<.01$) and insulin resistance (HOMA index) ($r = 0.76$; $P<.01$).
Conclusions: Increased plasma AOPP may contribute to the glucose, lipid metabolic dysfunction and insulin resistance in women with GDM and accumulation of placental AOPP may participate in the pathogenesis of PMPC. Further research about the relevance between the level of AOPP and the onset of PMPC in GDM was needed in order to have a profound prospective in oxidative stress

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P1.17.
SEXUAL DIMORPHISM STARTING FROM THE BLASTOCYST STAGE IN RESPONSE TO AN IMBALANCED MATERNAL DIET IN A RABBIT MODEL

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Maternal environment during early developmental stages has been largely demonstrated to play a role in the establishment of adult phenotype in humans and animals. Using a rabbit model, we previously showed that feeding dams with a diet supplemented with high unsaturated fat and cholesterol (HH diet) from the prepubertal period and throughout gestation induced a metabolic syndrome in adult offspring. Here, we examined the effects of the HH diet on feto-placental phenotype at D28 post-coïtum (near to term) in relation to earlier effects observed in the blastocyst (D6). At D28, HH fetuses were intrauterine growth retarded and dyslipidemic, with males more affected than females. Lipid droplets accumulated in the trophoblastic layer of the HH placentas, consistent with the significantly increased concentrations in cholesteryl esters, triacylglycerol and fatty acids observed in D28 placentas. Total membrane and stored fatty acids concentrations were significantly higher in female compared to male HH placentas, whereas triacylglycerol was increased only in HH males. Lipid droplets were also observed in the trophoblast at the blastocyst stage. The expression of numerous genes involved in lipid pathways differed significantly according to diet both in placenta and blastocyst. Among them, *LXR-α* in HH placentas and *Adipophilin* in HH blastocysts were expressed in a sexual dimorphic manner. These data demonstrate that the maternal HH diet induces sex-dependent metabolic adaptations of the trophoblastic layer from the blastocyst stage until term that appear to protect female conceptus from developing severe dyslipidemia compared to male conceptuses.

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P1.18.
MATERNAL OBESITY: GENDER-SPECIFIC DIFFERENCES IN PLACENTAL PATHOLOGY

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Background: Adverse effects of obesity have been linked to inflammation in various tissues, including adipose tissue and pancreas. However, studies on placental inflammation and obesity have demonstrated conflicting findings. We sought to investigate the relationship between maternal obesity, the associated complications diabetes and hypertension, and chronic villitis (CV).
Design: Patients with singleton, non-anomalous pregnancies delivering after 35 weeks were included. Obesity was defined as pre-pregnancy BMI ≥ 30 and normal weight as a BMI between 20 and 24.9. Covariates included diabetes, defined as either Type 2 diabetes or gestational diabetes requiring medication (GDMA2), and hypertension, defined as chronic or gestational hypertension or preeclampsia. Data was analyzed using Chi square and logistic regression; odds ratios and 95% confidence intervals (CI) were calculated.
Results: 213 obese and 177 normal weight patients were included in this analysis. Diabetes and hypertensive disease were associated with obesity (52.6% and 52.1% vs. 6.2% and 16.9% in the obese vs. normal weight population, respectively, $p<0.001$). CV was also more common in the obese group (25.4% vs. 16.9%, $p=0.044$); however, neither diabetes nor hypertension was associated with CV. Among obese patients only, univariate analysis revealed that CV was 1.8 times as prevalent in the placentas of female neonates (33% female vs. 18.2% male, $p=0.013$). Obesity [OR=1.701 (1.02, 2.81)] and gender [OR=1.6 (1.03, 2.77)] remained associated with CV in the multivariate model.
Conclusion: The effect of obesity on inflammation, as manifested by CV in the placenta, is independent of diabetes and hypertension, but is significantly associated with fetal sex, with a higher frequency of CV in pregnancies with female fetuses. Since CV is associated with fetal growth restriction, it is possible that CV may influence fetal growth in the setting of maternal obesity. These data underscore the importance of placental histopathology in evaluating effects of maternal obesity on pregnancy outcome.

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P1.19.
THE G PROTEIN COUPLED RECEPTOR 55 IN THE HUMAN PLACENTA AND THE ROLE OF ITS ENDOGENOUS AGONIST LYSOPHOSPHATIDYLINOSITOL (LPI) ON ENDOTHELIAL FUNCTION

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Objectives: The G protein coupled receptor (GPR)55 regulates many physiological functions, including vasorelaxation and angiogenesis. Here the spatio-temporal expression pattern in the human placenta was analyzed and the influence of its endogenous agonist lysophosphatidylinositol (LPI) on endothelial function was investigated.
Methods: For characterization of GPR55 expression immunohistochemistry (IHC), Western blot and PCR were performed on human first trimester and term placental samples and placental cells. The influence of LPI